〔Clin. Chim. Acta, **201**, 201-206 (1991)〕 [Lab. of Molecular Biology]

Anti-skeletal Muscle and Anti-acetylcholine Receptor Antibodies in Patients with Thymoma without Myasthenia gravis: Relation to the Onset of Myasthenia gravis.

MITSUHIRO OTA, MOTOHIKO ITO, HIROKAZU HARA, NOBUYUKI ITO,
HIROSHI NISHITANI, KYOZO HAYASHI*, KIYOE OTA

We measured the anti-skeletal muscle (SM) antibody titers in sera from 46 patients with thymoma but without Myasthenia Gravis (MG) in order to determine whether the presence of anti-SM antibody is linked to the combination of thymoma-MG, or to thymoma alone. We detected anti-SM antibodies in 18 of these sera, of which 15 had concomitantly elevated titers of anti-AChR antibodies. Moreover, 9 of whom had experienced the onset of MG after surgery. In contrast, no patient without elevation in both antibodies developed MG during the followup. We concluded that the presence of anti-SM antibodies is linked strongly with thymoma associated with MG.

〔Clin. Chem., **37**, 36-39 (1991)〕 [Lab. of Molecular Biology]

Carbonic Anhydrase III in Serum in Muscular Dystrophy and Other Neurological Disorders: Relationship with Creatine Kinase.

MITSUHIRO OTA, YASUKO INAGAKI, NOBUYUKI ITO,
KYOZO HAYASHI*, HIROSHI NISHITANI, KIYOE OTA

We measured with a radioimmunoassay the concentrations of carbonic anhydrase III in sera from 68 patients with muscular dystrophy, 10 carriers of Duchenne muscular dystrophy (DMD), and 63 patients with other neurological disorders. The values obtained were compared with those for creatine kinase. Serum CA-III was strikingly increased in patients with DMD (mean, 274.4 μg/L) and congenital (Fukuyama-type) (182.8 μg/L) and limb-girdle (203.7 μg/L) dystrophies and positively correlated with the activities of CK in patients with DMD. CA-III concentration decreased with the subjects' age and severity of the disease.

〔Biochem. Biophys. Res. Commun., **175**, 215-219 (1991)〕 [Lab. of Molecular Biology]

Memory Impairment and Morphological Changes in Rats Induced by Active Fragment of Anti-nerve Growth Factor-antibody.

TOSHITAKA NABESHIMA, SHIN-ICHI OGAWA, HIROHISA ISHIMARU, TSUTOMU KAMEYAMA,
TANEO FUKUTA, RIE TAKEUCHI, KYOZO HAYASHI*

Treatment of rats with a specific Fab' fragment of anti-nerve growth factor (NGF)-antibody (anti-NGF, 12, 120, and 400 μg/4 weeks, i.c.v.) impaired their learning ability. The distance of swimming of anti-NGF-treated rats in a water maze was shortened more slowly by training than that of control rats. Anti-NGF treatment altered the staining of nuclei of cells in the hippocampus, parietal cortex and dentate gyrus with hematoxylin. It is suggested that the anti-NGF-induced amnesia could be due to change in nuclear morphology. This study is under progressing to make clear the relationship between NGF and the Alzheimer's disease.